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Pentachlorophenol and Contained Chlorinated Dibenzodioxins in the Environment

A Study of Environmental Fate, Stability, and Significance When Used in Wood Preservation

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Pentachlorophenol (PCP) like many other industrial chemicals, is under scrutiny by several government agencies. The issues of concern with PCP as a wood preservative are the environmental effects of impurities in technical grade PCP, and/or environmental effects of breakdown products of PCP, including health effects of these impurities or breakdown products as well as of PCP itself.

The U. S. Environmental Protection Agency (EPA), after studying the chlorinated dibenzodioxin impurity issue for several years, submitted the question of significance of PCP dioxin impurities to the EPA Science Advisory Board for review and recommendations in December 1975. In an EPA Office of Pesticide Programs paper given to the Science Advisory Board Executive Committee it was noted that over 80% of PCP produced is used in the wood preserving industry. The paper continued . . "Therefore, there is a question of the amount of dioxins which may leach into the environment and eventually reach the food chain. There also exist differences in opinions among the various manufacturers whether or not the dioxins found in PCP actually present an occupational and/or environmental hazard". Pentachlorophenol was one of twenty-five pesticides included in another detailed study of the President's Council on Environmental Quality, which is intended to evaluate, among other things, environmental impact of toxic substances. The National Institute of Environmental Health Sciences (NIEHS) sponsored a conference on chlorinated dibenzodioxins and dibenzofurans at Research Triangle Park, N. C. in 1973 at which there were several papers on pentachlorophenol and its impurities. Similarly, the American Chemical Society sponsored a symposium in Washington, D. C. in 1971 on Chlorodioxins—Origin and Fate. EPA and NIOSH (National Institute of Occupational Safety and Health) contractors, such as the Pacific Biomedical Research Center at Honolulu and Kettering Laboratories, University of Cincinnati, have been studying health effects of PCP for many years. These are only a few of the more visible investigations on the use of PCP in the environment and its effects in occupational health.

This heightened concern[1, 2, 3] was justified in the early 1970's due to the lack of knowledge about environmental impact of PCP and especially its contained chlorinated dibenzodioxin impurities. The use of PCP as a wood preservative has been steadily increasing to the point where over 48 million pounds were used in the U. S. in 1974. With this volume of usage it is important to know the environmental fate of these chemicals if they leave the wood by volatilization, leaching and migration, or when the wood is ultimately burned, chipped for animal bedding, or used in other applications. This paper is intended to answer many of the questions that have been raised over the past three years by EPA's Criteria & Evaluation Division, Office of Pesticide Programs, over the environmental fate of both PCP and its major impurities. These discussions and meetings with EPA were held at our request for the purpose of determining the data gaps which could present problems in continuing the registration and use of PCP as a wood preservative.

Brief History of Dioxin Issue

There are two separate issues that are to be evaluated—PCP as a toxic chemical, and the impurities in technical grade PCP. A discussion of the former will be deferred until later in this paper. Concerning the dioxin issue, there has been much confusion among the industrial users of PCP over the conflicting information.[4] One manufacturer of PCP considers

it necessary to remove most of the chlorinated dioxins and dibenzofurans from PCP in a new process which is claimed to have cost the company $900,000 in development effort.[5] The development of this low dioxin product was in response to concerns expressed by the Federal Government and the manufacturer about the presence of toxic impurities, including chlorinated dioxins in commercial grades of PCP.[6] On the other hand, there is no epidemiological evidence that commercial grade PCP is any more an occupational health hazard than the purer product when it is properly used in the wood treating industry or when workers are handling the treated wood. It is important for the reader to understand just what the dioxin issue is, how it relates to PCP, and to separate the dioxin issue from the subject of PCP toxicity.

Although there were prior publications about the potential hazards of chlorinated dibenzodioxins in 2,4,5-T and chlorophenols, including PCP[7, 8] and quiet research was going on within both the Food & Drug Adminstration and the U. S. Dept. of Agriculture in 1969, the dioxin question in PCP was exposed to the public by a hard-hitting article in the New Yorker on March 5, 1970 by Thomas Whiteside.[9] Whiteside wrote extensively about the hazards of 2,4,5-T here and in Vietnam, the fetus-deforming (teratogenic) effects of dioxin impurities in 2,4,5-T, the chloracne found in workers because of dioxins in 2,4,5-T manufacturing plants, and he also named PCP and other chlorophenols as chemical precursers of dioxins ("predioxins") under certain thermal conditions. He claimed PCP, when broken down by sunlight, forms chemical precursers of chlorodibenzo-p-dioxins. On June 25, 1970 John Dellenback (Rep.-Oregon) spoke in Congress before the House of Representatives and read into the Record[10] another article by Whiteside published in the New Yorker on June 27, 1970. This article, although again written on 2,4,5-T, mentions that PCP, a widely used wood preservative, can form dioxin when heated. He wrote, "Since the fate of most timber is to be burned sooner or later, and since it is reported that when five grams of pentachlorophenol is heated at a temperature of three hundred degrees for twelve hours it is capable of generating one and a half grams of octachlorodibenzo-p-dioxin, the possibility that considerable amounts of dioxin will be released into the atmosphere from wood treated with this preservative presents a potential health hazard of very alarming dimensions."

On November 5, 1970 Philip C. Kearney of USDA presented research results[11] before a joint meeting on pesticides, United Kingdom, Canada, U.S., sponsored by the Council on Environmental Quality and the President's Cabinet Committee on the Environment. This paper described the state of knowledge on dioxins. At that time the USDA Agricultural Research Service Biometrics Study had listed 18 compounds identified as chlorophenols or being derived from chlorinated phenol precursers that were currently registered pesticides that could conceivably be contaminated with chlorodioxins. (Earlier in 1970 the FDA had compiled a list of chick edema factors for dichlorophenol, trichlorophenol, and pentachlorophenol). The Biometrics study examined the measured amounts of tetra, hexa, hepta, and octachlorodioxins present, but this was not complete because there are theoretically 75 possible isomers of the dioxins and there were only six or seven synthetic isomers available as standards. These isomers vary widely in their toxicological characteristics. For example, at that time it was known that the extremely toxic 2,3,7,8-tetrachloro-dibenzo-p-dioxin (2,3,7,8-TCDD) has a single oral LD_{50} of 0.006 mg/kg in male guinea pigs while the 2,7-dichlorodibenzo-p-dioxin (DCDD) and octachlorodibenzo-p-dioxin (OCDD) have low acute toxicities. Doses as large as 1 to 4 g/kg of OCDD and DCDD did not cause death in rats.[12] Isomers of the same dibenzo-p-dioxin also vary in toxicological properties. While the 2,3,7,8-TCDD is highly embryotoxic and a potent acnegen, the 1,2,3,4-TCDD is neither embryotoxic nor acnegenic. Therefore, it is important to distinguish between the isomers as different, both chemically and in toxicological response. In U.S. commercial PCP the highly toxic lower chlorinated TCDD has not been found[1, 13, 14] utilizing analytical techniques capable of detecting 50 ppb. More recent information indicates its absence in PCP with analytical techniques capable of detecting 10 ppb.

Kearney reported[11] that TCDD is not taken up from soil by plants and is not translocated. It is immobile in soils and does not leach down into the soil profile. TCDD is photodecomposed under natural conditions, but slowly. Kearney also reported in 1970 that TCDD is a persistent compound, not easily broken down in soil.

Despite these potentially reassuring findings on TCDD, considerable activity on dioxin research was underway due to the October 29, 1969 press release disclosure by the Office of Science and Technology that 2,4,5-T caused deformed offspring when fed in large doses to mice and rats. The disclosure that this powerful teratogen, TCDD, was present in a widely known herbicide triggered a number of administrative and legal actions. Senator Philip Hart's Subcommittee on Energy, Natural Resources and the Environment conducted hearings in April 1970 on "Effects of 2,4,5-T on Man and the Environment." The Departments of Agriculture (USDA), Interior, and Health, Education and Welfare (HEW) jointly

announced suspension of home uses as a liquid formulation and all uses in and near waters, followed in May 1970 by cancellation of all registered uses of 2,4,5-T for certain food crops. (Later, on May 7, 1971, a 2,4,5-T Scientific Advisory Committee to the EPA, whose members were selected from names supplied by the National Academy of Sciences, recommended restoring registration of 2,4,5-T with certain restrictions).

On January 15, 1971 USDA called a meeting of PCP producers, FDA and the Department of Commerce in Washington (EPA did not attend) to review the dioxin issue and arrive at a plan of action. At that time there was concern that there may be some TCDD present in the higher chlorinated dioxins and that the hexachloro dibenzo-p-dioxin (HCDD) was a serious problem, being highly toxic and present in PCP. It was also thought that OCDD remained in the environment for a long time and was not degraded by sunlight or soil.

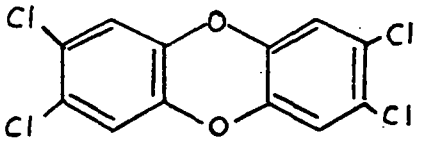
As mentioned earlier, the American Chemical Society sponsored a symposium on "Chlorodioxins—Origin and Fate"[15] in 1971 with 14 papers on dioxins. NIEHS sponsored a conference on chlorinated dibenzodioxins and dibenzofurans in April 1973 with 35 papers.[16] The EPA established a Dioxin Implementation Plan, formulated as a result of a Dioxin Planning Conference held in Washington July 25–26, 1974 among government, industry, and academia members. There continues to be accelerating research about the health and environmental aspects of the chlorodioxins and chlorinated benzofurans. There will be more information needed to make the important decisions—information on the subacute effects of dioxins, particularly the hexachlorodioxins; (1,2,3,6,7,8 and 1,2,3,7,8,9 isomers); information on metabolism, distribution and excretion of OCDD, HCDD and heptachloro dibenzo-p-dioxin. In several meetings held with EPA's Criteria & Evaluation Division, the following information gaps relating to PCP were defined:

1. Is dioxin in PCP treated wood in the same concentration as in the PCP prior to treatment?
2. Do the dioxins stay in the wood? Do they migrate to the surface of wood and into the environment? If so, how much?
3. If dioxins migrate to the soil or water, what is their environmental fate?
4. What happens when treated wood is ultimately disposed of by burning? Are dioxins formed, and if so, what type and amount?
5. If the low toxicity OCDD is exposed to the sun on the surface of wood, can it be dechlorinated to more highly toxic HCDD and TCDD? Can PCP degrade in sunlight to OCDD and ultimately to TCDD?

For the past four years Koppers Company has had a number of research projects underway to help answer these questions in order to provide information useful in retaining the registrations for PCP as a wood preservative. Much of this information is presented in this paper.

Significance of Chlorinated Dibenzo-P-Dioxins in PCP

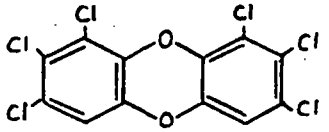
TCDD is the compound that has created most of the concern over dioxins due to its extreme toxicity (comparable to thalidomide but said to be 100,000 to one million times more potent in causing birth defects and the most toxic small molecule synthetic known). It has a structure,



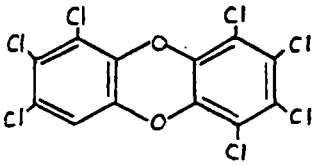
2,3,7,8 tetrachlorodibenzo-p-dioxin (known as 2,3,7,8-TCDD)

This dioxin has never been found in technical PCP produced in the United States, according to the literature.

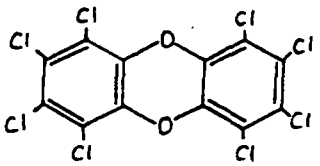
The chlorinated dibenzo-p-dioxins of interest in PCP are the hexa-, hepta- and octachloro dibenzo-p-dioxins, illustrated by the following examples of isomers:



1,2,3,7,8,9-hexachlorodibenzo-p-dioxin (1,2,3,7,8,9-HCDD) [one of ten possible isomers]



1,2,3,4,7,8,9 heptachlorodibenzo-p-dioxin [one of two possible isomers]

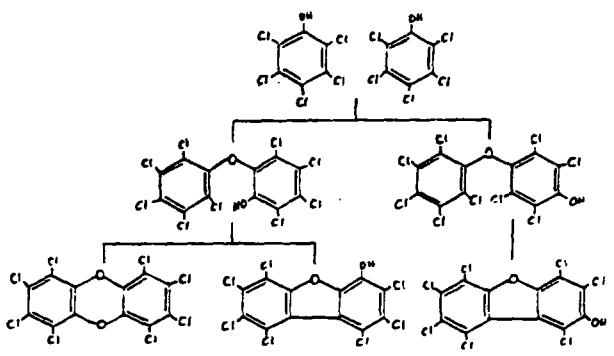


octachlorodibenzo-p-dioxin (OCDD)

The amounts of these impurities found in technical PCP can vary considerably depending on the analytical technique used and the particular sample of PCP. One manufacturer has used a technique in-

volving separation of dioxins from PCP by extraction of the PCP benzene solution with aqueous sodium hydroxide; a gas chromatograph equipped with an electron capture detector is used to detect and quantitate the chlorodioxins. The lower limit of detection varies from 0.1 ppm to 1.0 ppm depending on the dioxin. This method results in much higher total dioxin results from a PCP sample than the more accurate method of Gas Chromatography—Mass Spectrometry (GS–MS). For example, by using GC alone on a sample of PCP, we found 2800 ppm by weight of OCDD. Another laboratory found 2700 ppm. By using GC after ion-exchange chromatography on duplicate portions of the same sample, we found 1100 ppm OCDD. Other contaminants in PCP such as the hydroxychlorodiphenyl ethers and octachlorodibenzofuran, which is present in PCP, cannot easily be separated from OCDD by GC techniques alone. Also, another chemical intermediate, 2,3,4,5,6,6-hexachloro-2,4-cyclohexadienone is also found as a contaminant that elutes in the gas chromatograph at 0.2 min. less than OCDD. It converts to 2,3,4,4,5,6-hexachloro-2,5-cyclohexadienone by heating at 210°C. Both interfered with GC analysis of OCDD and tended to increase the apparent amount of OCDD.[17, 118]

There is another more significant analytical problem that has been recognized. The analytical equipment used can, through heating the sample, generate dioxins that were never in the sample before analysis. The hydroxychlorodiphenyl ether described above is known as a precurser of dioxin. The reaction has been described[18] as two molecules of PCP reacting with each other under alkaline conditions in two steps to form OCDD. The first step, forms the 2-hydroxynonachlorodiphenyl ether called a "predioxin." If a chlorine in the para or meta position to the hydroxyl group in one molecule reacts with the hydroxyl group of another molecule, formation of the dioxin is hindered. Jensen and Renberg[18,19] using thin layer chromatography, ion exchange, and GC–MS found that a PCP sample containing 1100 ppm of OCDD actually contained only 50 ppm OCDD when the "predioxin" in the PCP sample was prevented from forming OCDD in the GC column. They used diazomethane to methylate any remaining phenolic compounds in the base insoluble fraction and the polychlorohydroxy diphenyl ethers, which are sterically hindered, reacted and were methylated, making it impossible for them to form OCDD. Thus, the high value of 1100 ppm OCDD was the sum of OCDD in the sample and the OCDD formed by heating in the GC column. It is noted that alkaline extraction is used to prepare the sample for GC analysis and both heat and alkalinity favor ring closure of the "predioxin."[20] Therefore, indications are that analytical techniques sometimes used are causing the high level of dioxins found and the true values of dioxins in PCP are much lower. Also, the same reasoning applies to the hepta homologues.[19] The hexa dioxins were not investigated in this manner. It was noted, however,[19] that alumina column chromatography or concentrated sulfuric acid treatment of extract removes the "predioxins" and analytical error can then be avoided.



Other investigators[21] found that the heated inlet system of the gas chromatograph may have been responsible for the ring closure of the main impurity in PCP, 3,4,5,6-tetrachloro-2-(2,3,4,5,6-pentachlorophenoxy) phenol, to OCDD in the neutral fraction. These findings were discussed by Plimmer[1] in his paper at the Research Triangle Park Conference. Further study of "predioxins" was urged, but the point was made that PCP does have chlorinated dioxins and dibenzofurans in the base insoluble fraction. It is only the amount which is disputed. Firestone, et al[22] tested eight samples of commercial PCP and found from 0.17 to 38 ppm of hexachlorodibenzo-p-dioxin (HCDD) in all samples by gas-liquid chromatography with electron capture. The authors also found the hepta and octachlorodibenzo-p-dioxin, dibenzofurans with 4 to 8 chlorine atoms, and ethers with 4 to 10 chlorine atoms.

Plimmer, et al[23] examined 11 samples of PCP by MS. Sample collection for these 11 samples covered the years 1953 to 1970. Three samples collected in 1970 had from 0.5 to 37 ppm of HCDD. The heptachlorodibenzo-p-dioxin content of these samples ranged from 90 to 135 ppm. It is, however, characteristic of commercial PCP that the level of the hepta isomer is a factor of 10 or more lower than the octa isomer. Woolson, et al[13] reported on the examination of these same 11 samples of PCP. Seven had from 10 to 100 ppm of HCDD. Both OCDD and heptachlorodibenzo-p-dioxin content of four samples ranged from 10 to 100 ppm and of six samples between 100 and 1000 ppm. Johnson, et al[24,25] reported that 1973 commercial PCP has concentration ranges of 9–27 ppm HCDD and 575–2510 OCDD. No 2,3,7,8-TCDD was detected.

It is, therefore, obvious that the analytical methods used by the authors differ significantly and that some may be including impurities that are not dioxins at all. Woolson, et al[13] used concentrated H_2SO_4 in his cleanup followed by an alumina column separation and finally GC characterization. The results of Johnson, et al are based on methods described by Crummett and Stehl.[25] These authors remove the interfering chlorinated phenoxyphenols after alkaline extraction by the use of a nonaqueous ion exchange.

While it is clear that the level of dioxin impurities in PCP is variable, for the purpose of discussion it is assumed that HCDD does not exceed 100 ppm and OCDD does not exceed 2000 ppm with the hepta isomer not exceeding 200 ppm in current commercial U. S. PCP.

Toxicity of Chlorodioxins in PCP

It was already mentioned that the HCDD was the most toxic impurity in technical PCP but it really is no more toxic than PCP itself. It has been reported to be acnegenic and embryotoxic, but from 1000 to 10,000 times less toxic than TCDD.[12] The acute toxicity of HCDD is approximately 100 mg/kg in male rats.[12] The only sign of toxicity noted in acute studies with HCDD was loss of body weight.[12] This toxicity is not too different from PCP itself since PCP acute toxicity LD_{50} is listed[26,27] as ranging from 50 to 140 mg/kg in guinea pigs, 70 to 130 mg/kg in rabbits, and in rats from 27 to 80 mg/kg depending on the solvent used. In man the oral lethal dose is listed as 29 mg/kg in the U. S. Dept. of H.E.W. Toxic Substances List. Therefore, the HCDD impurity is no more toxic in a single dose than PCP itself.

The acute toxicity of OCDD is not significant because oral doses of 1000 mg/kg did not cause death in five female rats and 4000 mg/kg did not cause death in four male mice.[12] When OCDD was fed to rats at 100 μg/day feces contained 95% of the total dose and urine 4%. That retained by the body was confined to the liver, adipose tissue, and skin. After 49 days on a control diet, the amount retained had decreased to about 20% of the concentration present at 21 days.[28,29] Therefore, the body eliminates OCDD taken by ingestion. Another study on the chronic effect of OCDD[30] taken in the diet of mice at levels of 0.5% and 0.25% indicates that OCDD at the 0.5% level is probably toxic to the liver. No evidence of carcinogenesis attributable to OCDD was noted. The dose rate of OCDD at 0.5% level was equivalent to about 1250 mg/kg/day in mice and 200 mg/kg/day in rats (calculated from the author's data). From these data then it is obvious that the dioxin impurities in PCP present no acute toxicity danger greater than PCP itself.

Considering the chronic effects of HCDD such as acnegenic response and teratogenicity, there are data available[12] which again show that the levels of HCDD in PCP are so low that the toxicity of PCP is the controlling factor, not the chronic toxic dose of HCDD. The HCDD is teratogenic in the pregnant rat at a 100μg/kg/day dose level given orally on days 6 through 15 of gestation. The no-effect level for embryonal or fetal development was between 0.1 and 1.0 μg/kg/day. As previously mentioned, OCDD was not teratogenic at 500 mg/kg/day, but it was embryotoxic at this high level. By using these known toxic doses of the impurity, HCDD, one can calculate the amount of PCP ingestion or absorption necessary to cause chronic toxic effects from HCDD. If the typical commercial PCP had the assumed maximum of 100 ppm HCDD and knowing the toxic effects, the commercial PCP doses calculated are:

1,000 mg/kg/day for teratogenicity
10 mg/kg/day for "no effect" level

Obviously, the lethal acute dose of PCP is the controlling factor. The known teratogenic chronic dose of HCDD could not be reached from PCP without death occurring from PCP itself. However, one must examine the significance of the "no effect" dose also. A person is assumed to inhale 10 cubic meters of air per eight-hour day. The maximum amount[31] of PCP in air that a person should be exposed to daily is 0.5 mg/M.[3] This level has been shown by industrial plant experience to be about one half the upper limit an uninitiated person can tolerate.[31] PCP dust has excellent warning properties, such as painful irritation, sneezing, and coughing before one is subjected to concentrations which will produce any adverse systemic effects. Therefore, one would have to tolerate painful exposure an entire day in order to reach the "no effect" level by breathing PCP dust. In Russia,[32] continued exposure to a level of 0.32 mg/M[3] PCP caused symptoms in workers of headaches, tiredness, burning of the eyes, nose, mouth and skin for 3 to 5 hours after exposure. These workers had PCP on the skin of their faces, necks and hands in quantities of 0.56 to 3.26 mg/sq. cm. It is hardly likely that current industrial hygiene practices would permit exposure to continual concentrations of dust of this magnitude. Even if such exposure occurred, the "no effect" level for embryonal response to HCDD would not be reached because a 120 lb. person's "no effect" level of HCDD requires from 54 to 540 mg/day of PCP. This is 10 to 100 times the amount he could inhale from breathing dust. Therefore, again the toxic effect of PCP it-

self is the controlling factor, not the chronic effect of low level doses of HCDD.

It is not likely that the absorption of PCP from a person's skin contact with liquids or from ingestion could approach the lower range of the "no effect" level (54 mg/day, equivalent to HCDD of 0.1 μg/kg/day) without seriously affecting his health and well being. This statement is based on the knowledge that PCP is rapidly eliminated from the body via the urine so the concentration in the urine would be about 20 to 30 mg/l. However, case histories[33,34] have shown illness from PCP when the concentration in the urine was 10–30 mg/l. From this interpretation of the toxic effect of HCDD and its significance, it is apparent that the relatively high level of HCDD assumed, 100 ppm, will not produce toxic effects from PCP exposure. In fact, levels of HCDD in commercial PCP today do not exceed 30 ppm.[24]

Another factor that must be considered is the acnegenic effects of HCDD. Johnson, et al[24] wrote that the presence of HCDD in PCP gives rise to concern because the "product literature" warns that frequent skin contact may result in an acneform dermatitis. However, the authors report that "documentation of such occurrences is unavailable". There are reports in literature of chloracne developing in industrial workers, but these reports usually have to do with exposure to other chemicals such as TCDD,[35,36] 2,4,5 trichlorophenol[37] (by caustic hydrolysis of 1,2,4,5 tetrachlorobenzene where TCDD was the culprit), chlorinated naphthalenes, and chlorinated biphenyls.[38]

In Germany the production of pentachlorophenol by the alkaline hydrolysis of hexachlorobenzene resulted in reported cases of chloracne,[41] but it was recognized that either PCP itself or an intermediary product in its production could have been the causative agent; in addition, some of the men had been exposed to tetrachlorobenzol and trichlorophenol (technical trichlorophenol can have the TCDD present). It should also be noted that today's American industrial hygiene standards are considerably above those of European plants, especially as compared to several years ago in Europe. These reports of chloracne resulting from exposure to chlorophenols were all relating to the manufacture of the chemical—not the use of the chemical where exposure levels are generally much lower.

Chloracne is a disease described by the formation of comedones with or without cysts and pustules. The follicular orifices are filled with sebaceous and keratinous material. Melanosis and a secondary inflammatory reaction may exist. Chloracne may result from direct contact (absorption) with the chemical producing it[39] or from systemic absorption[40] including inhalation.

The threshold for acnegenic response of HCDD was measured by Schwetz, et al[12] in a rabbit ear bioassay. Solutions of 10μg/ml in chloroform and dimethoxyethane produced positive response as indicated by comedones. This amount of HCDD in solution is equivalent to 10 mg/l of HCDD or 330 g/l of PCP (assuming a maximum level of 30 ppm HCDD in PCP). This is a concentration of PCP in solution of about 33%. Obviously, workers handling these concentrations should avoid skin contact with the solution, not only to prevent chloracne, but because these high concentrations (or any treating solutions) of PCP are quite toxic and can be absorbed through the skin. However, it is apparent that concentrations of PCP from 5 to 7.5% as used in the treating industry and as present in treated wood are well below the HCDD acnegenic threshold level. It should be noted that the acnegenic response to HCDD (and calculations based on it) cited above[12] was the result of tests with chloroform solutions. Later, however, it was shown[17] that the acnegenic response was probably greater due to the chloroform solvent than it would have been if a solvent were used that did not cause papules, desquamation and thickening of the tissue by itself. Besides chloroform causing these skin irritation problems, it has also recently been reported by the National Cancer Institute that mice and rats given high doses of chloroform develop liver cancer and kidney cancer. Thus, it appears that chloroform was a poor choice for a solvent for the rabbit ear tests.

Chick edema disease during the late 1950's and early 1960's was shown to be caused by toxic substances in the feed of chickens in certain feed grade fats but these were not due to PCP contamination alone. The disease is characterized by the presence of excessive fluid in the heart sac and abdominal cavity of chicks. These and other symptoms such as subcutaneous edema and liver necrosis are accompanied by high mortality beginning about the third week. The by-product feed grade fatty acids were obtained from production of oleic and stearic acids that were derived from tallow produced from stripping hides in the leather tanning industry. According to Firestone[8] and Metcalfe[42] the source of the chick edema factor was fleshing grease from hides that had been treated with pentachlorophenol and trichlorophenol in glue emulsions used for dry rendering. Cantrell, et al[43] showed that one of the active crystalline materials isolated from the toxic fat was HCDD. This was later shown to produce chick edema.[8,12] However, the toxic fat that caused the chick edema disease also contained TCDD from trichlorophenol that was used in hide soaking operations.[8] TCDD

is ten times more active than HCDD as a chick edema factor.[12]

There is no doubt that HCDD can cause chick edema, but OCDD does not.[12] Chicks receiving 10 to 100 μg/kg/day of HCDD administered orally as a corn oil/acetone solution, produced a positive response for chick edema factor.[9] Those receiving 100 μg/kg/day did not survive. Chicks receiving as much as 0.5% OCDD in their food had no effect.

The significance of these results in relation to wood preservation uses of PCP must be examined in terms of exposure. Obviously, PCP used in wood preservation is diluted and applied in a solvent to wood, either by pressure treatment or surface application. The most obvious means by which chicks can ingest PCP from treated wood is by the unrecommended use of bedding material containing sawdust from PCP treated wood. The "no effect" level for chick edema for HCDD is 1.0 μg/kg and for the first significant level is 10 μg/kg. If treated wood sawdust contains an average of 0.5 pcf and the PCP contains 20 ppm of HCDD, then it is possible to estimate the amount of sawdust a chick would have to ingest in order to reach the "first significant effect" level. If a chick ingests 18 g. of feed per day and averages 122 grams in weight over the first three week period, chicks would have to consume 30% of their diet as PCP treated sawdust to reach the "first significant effect" level from HCDD. These calculations were based on the toxic effect of a corn oil/acetone solution of HCDD which would be readily absorbed. Actually in sawdust PCP would probably pass through the chicks' system intact in the sawdust, thereby increasing the dose of HCDD necessary for chick edema factor.

Whether or not the use of PCP treated wood is free of chick edema factor, the use of PCP contaminated chicken litter is not recommended because a "musty" taint in chicken eggs has been shown to be caused by 2,3,4,6-tetrachloroanisole, which was present in the chicken litter. The source of the chlorinated anisole was the biological breakdown of pentachlorophenol and tetrachlorophenol by molds such as Aspergilli, Penicillium and Trichoderma species.[44, 45] Chickens containing as little as 0.02 μg/g of tissue were considered inedible due to "musty" taste. Also, no tolerances have been established permitting any residues of PCP or the chloroanisoles in the edible tissues of animals. Based on these reasons alone, PCP treated residue should not be used for chicken litter.

Significance of Toxicity of PCP—Human

There will be no attempt here to review the literature on toxic effects of PCP. Others[26, 27, 46, 47, 48] have done adequate jobs in this regard. When adequate precautions are taken PCP and its solution are safe to use in treating wood, but when adequate precautions are not taken acute and chronic toxicity can occur. It is sufficient to say that PCP is rated as a highly toxic substance following ingestion or skin penetration. There are at least 50 cases of chronic intoxication reported in medical literature where PCP exposure from industrial use was the cause. Skin penetration from dip solutions has been reported as the most usual route of absorption in these cases. For example, in Australia, workers spraying the solution (sodium pentachlorophenate) for herbicidal use on pineapple worked with unprotected lower limbs and often without a shirt. There were five fatal cases of poisoning reported.[49] In these cases the workers used concentrations of about 1 to 2.5% Na PCP in a water/mineral oil emulsion. In one case the concentration by mistake was 14% and the overalls were soaked in fluid. In Canada there is reported one death and four illnesses, all from one millwork plant where workers were dipping wood with bare hands.[50] Similar instances are reported in other literature[27, 46] where gloves and other protective clothing and adequate industrial hygiene practices were not used. The precautions and warnings shown on the label and in the product literature must be followed for safe use of all preservatives.

The chronic (long term) toxic effects of PCP must also be considered, not only in man, but in animals that might be exposed. PCP can enter the body through the skin without apparently damaging it, can be inhaled, and can be ingested in the form of dust (abrasion planer dust or sawdust). However, when workers are protected from overexposure and practice good personal hygiene, there have been no significant health effects from long term exposure.

In a study of worker exposure by the Environmental Health Sciences Center, Oregon State University, air samples and urine samples were taken at 25 companies using PCP as a wood preservative. The industrial exposure to PCP in these plants was well below the safe level. There were 7 companies dipping in wood with a 5% PCP solution, 11 using PCP for spraying rough sawn lumber for stain control, and 7 pressure treating plants. The concentrations of PCP in the air around the workers was found to be well below the TLV of 0.5 mg/M³ in all of the mills monitored. In the pressure treating plants the TLV was approached or exceeded only when the air next to the door was sampled during the short time immediately after the retort was opened. (It should be noted that the volatility of PCP with steam at 100°C is 0.167 g of material/100 g of steam at standard atmospheric pressure. In addition, there may be an oil mist inside the retort after final vacuum).

TABLE 1.—Air and Urine PCP Concentration from Plants and Mill Workers in Oregon.

Type of Operation	PCP in Air (mg/M³) Mean (range)		Urine PCP (ppm) Mean (range)
	Average Worker Exposure	Maximum Worker Exposure	
Dip	0.019 (0.003–0.063)	0.019 (0.006–0.063)	2.83 (0.12–9.68)
Spray	0.006 (0.003–0.012)	0.026 (0.004–0.069)	0.98 (0.13–2.58)
Pressure	0.014 (0.004–0.028)	0.297 (0.043–1.000)	1.24 (0.17–5.57)

The PCP concentration in urine varied considerably according to the type of treatment used in the plants as expected because the exposure varied. As shown in Table 1, the "average worker exposure" is the concentration found in the area where a worker spends the majority of his time, and the "maximum worker exposure" is that found in the maximum exposure area near the PCP source.

The urine concentrations found in this study agree well with those found by Bevenue, et al.[47] in a study of PCP applicators in Hawaii. In the University of Hawaii study[51] it was demonstrated that respiratory tract absorption is a reasonable explanation for the occurrence of PCP in the urine. The excretion half time of 10 hours confirms the rapid excretion of PCP from the body. However, there appears to be a delay between inhalation exposure and the excretion via the urine. Despite the rapidity of excretion, the failure of PCP concentration to return completely to normal even after long absence from exposure, suggests the binding of some PCP to the plasma protein or to the respiratory tract (tissue binding), delaying final excretion for several days. Detoxification of PCP by the body may also occur via methylation to the anisoles or conjugation with sulfate. It is known that this does occur in fish.[78] In mammals the PCP has been found to be metabolized to the breakdown products, pentachlorophenyl beta-glucuronide and a small amount of chloranil in the urine, intestines and liver.[52]

Long term chronic effects of low dose PCP exposure, however, appear to be absent. The University of Hawaii, Pacific Biomedical Research Center at Manoa has had an Epidemiologic Studies Program underway since 1967. In a prospective study of an occupationally exposed population annual physical examinations, blood chemistry and urinalysis testing were undertaken on all control and occupationally exposed adults. There were 21 workers having a mean of 3196 total days of exposure to PCP in an occupation of treating wood (pressure treatment). Although PCP was used by 70 other workers, such as pest control operators, the wood treaters had significantly higher levels of PCP than all other participants in the study. The mean blood serum level in the 21 workers was 1.05 ppm whereas the control was about 0.1 ppm. While vertigo and insomnia occurred more frequently among workers with high serum PCP levels, in tests of workers for neurological and psychological effects, there were no effects noted from PCP residue levels. In fact, PCP was the only pesticide of the four studied (PCP, Dieldrin, DDE, and DDT) that showed an insignificant contribution in a regression study. In comments on these long term studies, Bevenue[117] recently wrote, "no authenticated cases of illness or disease relationships have been observed or recorded during the local long-term study conducted in this subject area; we are referring to the period of time from 1967 to the present date, and to the local population both non-occupational and occupational (wood treating plants and commercial control operators) exposed in the PCP residue level range of 0.1 to 10.0 parts per million."

A urine analysis of workers in one of our PCP using plants was recently done on four workers who were occupationally exposed. Penta concentrations ranged from 0.05 to 0.34 ppm in these workers. As can be seen in Table 2, these values fall well within the ranges found for non-occupationally exposed persons in Hawaii.

The method of analysis used was substantially the same as that used by Bevenue. There is a reasonable probability that many of the reported values are high because of the presence of interfering substances which indicate as PCP by GC but can be excluded by GC-MS. This will be discussed in more detail later.

Another significant study of chronic effects of PCP in the wood preserving industry was reported by Bidstrup, et al.[63] in 1969 in England. The authors

TABLE 2.—Penta in Urine of Non-Occupationally Exposed Persons.[88]

Number of People	Number of Analyses	Conc. "PCP" in Urine, ppm Range	Mean
117	267	0.003–1.84	0.040
173	173	0.003–0.570	0.044

report that while most pesticides, including PCP, have been said to cause peripheral neuropathy, one company employing 1853 men working full time on the application of PCP wood preservative reported no cases of peripheral neuritis over a 10-year period.

Another study of chronic effects of PCP to cotton field workers in Russia[29] showed that concentrations in air as great as one tenth the threshold (0.32 mg/M^3) caused no health effects in workers. Animal tests that were carried out to determine cumulative effects of inhalation at 1/10 to 1/5 the LD_{50} dose showed it to be only slightly cumulative in effect.[29] Thus, the worldwide literature indicates no chronic effects from long term worker exposure.

The use of PCP in the wood preservation industry has not caused unusual health problems from PCP or dioxin impurities in plants operated by the Koppers Co. A detailed review of the medical records at each of our 28 wood preserving plants was made for the period 1961–1971. This survey covered approximately 33,400,000 working man hours (over ten years) based upon 1670 employees (annual basis). There were 19 plants using PCP in which 1,330 employees worked (annual basis). Over the ten-year period there were only 26 reported cases of PCP related health problems, all of which were attended by a physician:

chemical conjunctivitis	12
skin burns	9
allergy	1
dermatitis (arms and hands)	2
miscellaneous*	2
Total	26

* One case of gastritis from inhalation and one case of folliculitis on legs from continuously wearing trousers contaminated with PCP dust. This was not diagnosed as chloracne by the attending physician.

All these health problems were conditions caused by PCP itself and are not believed to be related to the dioxin content of PCP. If chloracne or other serious health effects would have been caused by PCP exposure, there certainly would have been more than 26 health effects in 1330 employees over a ten-year period. This health record, by itself, supports the logic and reasoning arrived at by the mathematical calculations above.

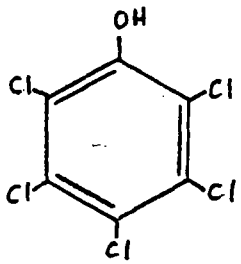
Nevertheless, despite these favorable use histories, researchers continue to speculate about the possible hazards of low level doses of PCP. At the American Chemical Society's 170th National Meeting last year, Dougherty[54] reported that he had found that every person in a group of 60 students had a low amount of "toxic and persistent pentachlorophenol" in their urine at levels averaging 20 ppb. It was also reported that he found PCP in seminal fluid at levels as high as 70 ppb and an average value on five measurements of 50 ppb. Dougherty wrote that "these results may have significant implications for birth defects and genital carcinoma if they are confirmed by more extensive and detailed studies."

This, of course, is not the first time that investigators have found PCP in the urine of persons not occupationally exposed to it. Besides the Hawaii data reported in Table 2, the range found in Great Britain[55] was 2 to 11 ppb and in Japan[56] 10 to 50 ppb—all among the non-occupationally exposed persons and separated widely by different water and food supplies, diets, and industries.

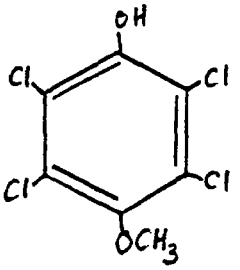
As to the allegations[54] of birth defects and cancer from low level doses of PCP such as one might get from occupational exposure, there is no basis for such fears. In fact, cancer researchers have found PCP to be non-carcinogenic in animal tests. Boutwell, et al.[61] tested the cancer promoting effect of various substituted phenols using dimethylbenzanthracene as the initiator and the phenol as the promoter in applications to the shaven backs of mice. While tumors developed with 2,4,5-trichlorophenol, phenol and substituted phenols with at least one unsubstituted position ortho to the phenolic group, there was no tumor promoting activity by PCP.[61] In these tests PCP was applied as a 20% solution in benzene for 15 weeks to 35 female Sutter mice. In 1969, a group of 13 investigators headed by J. R. M. Innes[62] found PCP to be negative as a carcinogen as a result of animal tests. They tested 130 compounds in two strains of mice, both male and female, by single subcutaneous injection and by continuous oral administration. PCP did not cause a significant increase in tumor incidence. The concentration of PCP in the feed was 130 ppm which was equivalent to 46 mg/kg body weight.

"PCP" Natural Background Level

Apparently, PCP, or a compound mimicking PCP in the analytical methods used, is ubiquitous in nature or else the clean-up procedures used in the analytical techniques are not good enough. The possibility of a naturally occurring "PCP" should not be ignored. In 1952 Anchel[57] reported that the antibiotic compound Drosophilin A, was para-methoxytetrachlorophenol ($CH_3OC_6Cl_4OH$) and was a metabolite of a fungus using potassium chloride as the sole source of chlorine. We have demonstrated that conventional gas chromatographic techniques using the electron capture detector will not easily distinguish between PCP and p-methoxy tetrachlorophenol. This is not surprising because these two compounds are nearly identical in molecular size and shape and have nearly the same retention times.



Pentachlorophenol
Mol. Wt. 266.35



P-Methoxy tetrachlorophenol
Mol. Wt. 261.94

Therefore, if p-methoxytetrachlorophenol (or some other contaminant) were present in the sample containing the unknown, it could easily be mistaken for PCP.

Contamination of samples must be carefuly avoided by the most scrupulous procedures in order to prevent parts per billion or parts per trillion of interfering substances from contributing to the observed value, which is also desired in the ppb range. The problems inherent in detection at these extremely low levels must be appreciated if one is to obtain meaningful values for toxic or environmental considerations. The natural background level of chemicals in the environment cannot be ignored.

The initial analytical method we have used for environmental studies of PCP involves the following steps:

1. A 5-gram sample of soil is extracted with a mixture of hexane and acetic acid in a Soxhlet extractor for approximately four hours.
2. The resulting hexane solution is extracted with aqueous alkali.
3. After acidification the aqueous phase is extracted with hexane.
4. The hexane solution is concentrated to a small volume by evaporation.
5. The PCP in the hexane is reacted with diazomethane to convert the PCP to its methyl ether.
6. The resultant solution is analyzed for PCP by vapor phase chromatography using an electron capture detector or by vapor phase chromatography followed by mass spectrometer.

Using these procedures many different samples of virgin soil and wood were analyzed—none of which were expected to contain any PCP. Some results are given in Table 3. Considering only the VPC results, there are a number of chemicals that can elute to give apparent PCP results, including tetrachloronaphthalene,[58] p-methoxytetrachlorophenol, 3-(3,4-dichlorophenyl)-1,1-dimethylurea (Karmex up to 20 ppb interference), and certain polychlorinated biphenyls. Because of the lack of specificity GC-ECD is useless in the ppb and ppt levels unless suitable precautions are taken to eliminate possible interferences. However, from Table 3 it can be seen that even with mass spectrometry there are unexpected background levels of PCP.

Despite our findings investigators continue to rely on GC methods for environmental research and report values of PCP in rivers such as the Willamette[89] in amounts ranging from 0.01 to 31 ppb. While Buhler, et al used mass spectrometry for final verification of PCP, their quantitative findings by GC-ECD could have been high due to other natural background materials which mimic PCP in the GC-ECD analysis.

Upon re-examining our technique[59] it was discovered that even with clean laboratory procedures, including heating glassware in a "burnout" oven operating at 500°C, somehow one to two micrograms of PCP were getting into the samples during the Soxhlet extraction step. This PCP came from the general laboratory atmosphere, apparently from the vapor phase. This procedural contamination was undoubtedly responsible for the PCP detected in "clean" samples, but the PCP was reduced to less than 1 ppb in the "clean" samples. Another source of contamination in the ppb and ppt range is the high purity analytical grade reagents used for the pH adjustment.[59, 60]

After accounting for the above analytical problems, we also found that municipal chlorination of drinking water can give rise to chlorinated phenols in the ppb range. Not only are the lower chlorinated

TABLE 3.—Comparative Apparent PCP Content of Samples Not Expected to Contain Any PCP—Vapor Phase Chromatography and Mass Spectrometry.

Sample	Apparent Pentachlorophenol, ppb	
	VPC	MS
Soil 1	63	47
Soil 2	9.6	18.7
Soil 3	43	39
Ponderosa Pine Wood	247	103
Douglas fir wood	113	8.5
Drinking water (fluoridated)	2.9	2.1
Humic Acid	2159*	
Tannic Acid	138*	
Rural water supply	2*	
Tea made w/distilled water	2*	
Southern yellow pine extract	6860*	
Ponderosa pine extract	560*	
Douglas fir extract	680*	
Western white fir	96*	

*These samples were preliminary work using bis (trimethylsilyl) trifluoroacetamide to make the derivative rather than using diazomethane to make the methyl ether derivative of PCP.

phenols formed, but a 10 ppm level of chlorine can chlorinate 1 ppm of phenol (from natural sources) and generate 0.2 ppb of PCP. The phenol in this case can be in reservoirs from leaves and other woody material. Although the amount of PCP formed is small it cannot be ignored when measurements are made in the ppb and ppt range. Dougherty[54] found PCP in the Tallahassee, Florida water supply at the level of 0.1 ppb, comparable to the level we found by chlorination of natural phenol. Nevertheless, it is difficult to account for the background level of "PCP" found in non-occupationally exposed persons by Dougherty[54] and others.[53, 55, 65] Either a naturally occurring process is responsible or the result is more likely due to contamination of samples or poor cleanup procedures.

Significance of Toxicity of PCP—Farm Animals

PCP has become very heavily used for preservation of fence posts, barn poles, and farm lumber which in itself is a testimonial as to its lack of significant health effects in farm animals. Surely, if PCP treated lumber caused health effects, they would become well known over the past 30 years. There have been several studies, however, to confirm these observations.

The most significant study, because it has to do with feeding animals doses of the treated wood rather than doses of solutions, is that of the New Zealand Wallaceville Animal Research Station.[64] Sheep and calves were fed doses of PCP treated wood. Results from early work showed that adult cattle were unlikely to be affected by PCP poisoning so cattle were not used in the study. For sheep, the minimum acute lethal dose was 145 g of treated Douglas fir wood—the wood sawdust containing about 1.1 pcf of PCP in a 5.56% W/V concentration in a petroleum distillate having the following properties:

specific gravity:	0.879
distillation range:	207 to 377°C
viscosity at 100°F:	38.4 SUS

The acute lethal dose of PCP for sheep was approximately 120 mg/kg, or about 3.6g of PCP, or 65 ml of preservative solution for a 30 kg sheep. Adverse cumulative effects from chronic feeding studies are caused by a lowest dose rate daily intake of 27.8 mg/kg. This was equivalent to 34 g of treated wood or 15 ml of solution (1.7 g of PCP) per day.

In the feeding of calves the acute toxicity lethal dose rate was 565 g of treated wood or 252 ml of preservative solution (14 g PCP) for a 100 kg calf. This is an LD_{50} of 140 mg/kg. Chronic feeding studies showed that daily doses of 143 to 204 g of treated wood would be necessary to eventually cause death from chronic poisoning in a 100 kg calf. This is equivalent to 63 to 90 ml of solution or 3.5 to 5 g of PCP. The chronic LD_{50} is 35 to 50 mg/kg PCP.

Harrison[64] concluded, "Considering that the acute fatal quantities must be ingested at one time and the chronic quantities regularly every day, it is extremely unlikely that stock could ever obtain injurious amounts by chewing treated woodwork or through licking the surface of wood properly treated." It should be noted that the fairly high retention of 1.1 pcf in the wood fed to the animals was supposed to simulate the high retentions near the surface of treated wood.

Despite the relative safety of treated wood, the solutions themselves can be quite dangerous to farm animals if they should ingest a dose accidentally.[64] Spencer[65] reported the death of a Hereford cow which had ingested a 5% solution of PCP in kerosene, although it is almost certain that the kerosene alone would have killed. Walters[66] orally drenched swine with 30 g of PCP solution (5%) and sheep with 8 to 35 g of PCP solution (5%). Drenched animals were slaughtered and chemical analyses and histological examinations were made on the tissues. None of the livestock showed any visible effects resulting from the drenching experiment, but moderate cell degeneration was noted in the kidneys and liver of swine (not enough to cause death). No harmful effects were noted in the kidney or liver of the ewes or the kidneys of the lambs; some detrimental effect was noted in the liver of lambs. These experiments and other feeding studies on animals show that while PCP is toxic to living things, it can be ingested in quantities less than threshold and be rapidly eliminated without any significant effect.

There are cases where PCP can be extremely toxic to young swine. Toxicosis is particularly evident in swine having extended direct contact with freshly treated lumber containing excessive quantities of PCP in heavy oil. The oily wood causes sufficient skin absorption to cause fetal deaths in the pregnant sow and/or weak pigs at birth. Sufficient bedding can avoid this problem, but dry or non-bleeding lumber can prevent it entirely. Direct contact by the lactating sow with freshly treated lumber will discourage nursing due to PCP on the teats and mammary glands or if the piglet does nurse, PCP is ingested. In either case mortality by either starvation or toxicity can result. Again, proper selection of the diluent and choice of dry, non-bleeding lumber is essential to avoid these problems.[67, 68, 69] Also, aging of the lumber is needed as well as sufficient bedding to prevent direct contact of the body of the sow with the floor of the farrowing pens.

Toxicity to Fish

Pentachlorophenol and its sodium salt are fatal to the more sensitive species of fish in concentrations above 0.2 ppm although hardier species will survive at 0.4 or 0.6 ppm.[70] The toxicity is increased by lowering the pH of the water. Apparently, the PCP reaches the site of action via the gill parts. Eggs of lake trout are very resistant to PCP, but lake trout are most sensitive to PCP in the yolk sac stage immediately after hatching.[70] Guppies *Lebistes reticulatus* can become acclimated by sublethal levels (1 ppm) of PCP and survive at more toxic levels.[71] One of the most sensitive fish to PCP is the salmon. The growth rate and food conversion efficiency in underyearling sockeye salmon were almost equally affected by exposure to sodium PCP, with EC_{50} values about 1.80 ppb for each measure of toxicity. The 96-hour swim performance LC_{50} (lowest concentration—50%) was 63 ppb. Apparently, concentrations of 50 ppb do not affect swim performance.[72] In fact, work at Oregon State University Environmental Health Science Center[73] with juvenile chinook salmon and stream invertebrates suggests that salmon production is not significantly affected in streams containing PCP at concentrations of 6 ppb (or one tenth the 96 hr. LC_{50}).

Recent work reveals that although fish can concentrate PCP in the gall bladder[80, 81] they excrete it, mostly in a detoxified bound form identified as pentachlorophenylsulfate.[78] This conjugate is identical to that found in the detoxification mechanism of PCP by the short necked clam.[82] The observation in laboratory tests has been that the PCP concentration gradually decreased in the media during the culture of fish such as rainbow trout[83] and carp,[84] but the amount of PCP accumulated by the fish was smaller than that lost from the media. It is probable that the decrease was due both to photolysis (degradation by light) and detoxification by sulfate conjugation in the body of the fish.

From the above discussion it is apparent that while PCP is toxic to fish, there is also a detoxification mechanism occurring naturally by biodegradation, conjugation with sulfate by shellfish, degradation by light, and methylation.

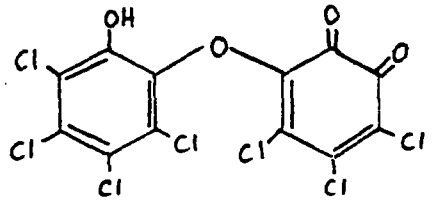
It is well known in the wood preserving industry that fish kills can easily occur if there is seepage into streams of contaminated effluent or spills of PCP solution. Considerable investment has been made in ways and means to prevent such an occurrence.

It is unlikely that PCP could contaminate bodies of water from treated wood such as stacks of lumber treated with sap stain control chemicals or treated poles or posts installed. The reason for this is that PCP is biodegraded by soil when in very low concentrations, and it is easily degraded by sun when exposed in water. Any PCP that leaves the surface of wood via rainwater, reaching the soil and eventually entering a body of water would have a short life if exposed to the sun.

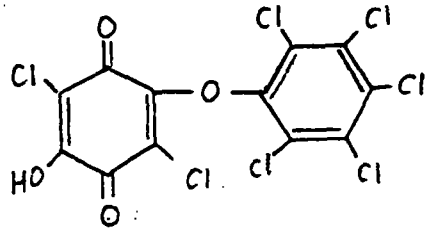
The Stability of PCP and Chlorinated Dioxins to Sunlight

In the use of treated wood, the PCP on the surface of the wood is subject to the degradation effects of sunlight. Light will cause photodegradation (photolysis) of both PCP and the chlorodioxins to harmless byproducts, especially if water is present. After a relatively short time the exposed surface has had a considerable reduction of dioxin and PCP content, as explained below. Thus, unless oil solution continues to migrate out from underlying layers of wood, the wood surface decreases in toxicity as it weathers. Nevertheless, the degradation by sun and combustion has been regarded by some environmentalists[9] to be cause for alarm since theoretically the highly chlorinated dioxins could dechlorinate to more toxic dioxins, and the PCP could theoretically form OCDD on heating[8, 74, 75, 76, 77] or when exposed to sunlight.[90, 91] There has been considerable research done on this question to determine what the mechanisms are and what the degradation products are.

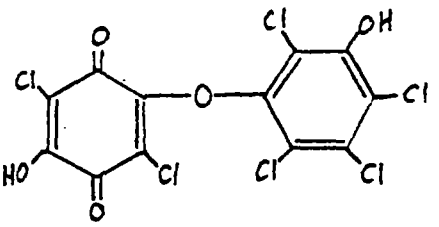
In 1966, Kuwahara, et al.[85, 86] identified the main products of photochemical breakdown of the sodium salt of PCP in water by sunlight to be chloranilic acid and a yellow compound, 3,4,5-trichloro-6-(2'-hydroxy - 3',4',5',6'-tetrachlorophenoxy) - o - benzoquinone.



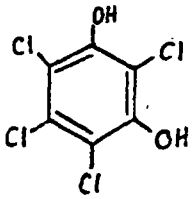
The minor products were a red compound,



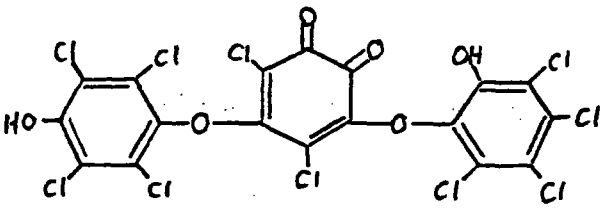
and an orange-red compound,



Haitt[87] and Munakata, et al[88] also reported that additional breakdown products of Na PCP in water by sunshine were tetrachlororesorcinol



and a yellow compound formed from an intermediate and tetrachlororesorcinol.



All of these breakdown products had weaker fish killing activities than Na PCP. Interestingly, these researchers did not identify any dioxins from the photolysis of Na PCP in water. In contrast, Plimmer, et al[90] identified OCDD and the heptachloro dibenzo-p-dioxin in the irradiated solution of aqueous sodium hydroxide/pure dioxin-free PCP. However, the maximum amount that could be generated from a solution containing 1000 ppm of Na PCP was 36 ppm of OCDD and a smaller amount of the hepta dioxin. This very small yield was not reproducible. No TCDD was formed. However, traces[91] of hepta and hexachlorodioxins were formed from photoreduction of OCDD *when dissolved in alcohols.* Under these conditions (presence of a hydrogen donor) any TCDD formed is photodecomposed very rapidly—more rapidly[91] than it could be formed from OCDD. For example, as seen in Figure 1,[92] the photodecomposition rate of TCDD in methanol was 2.3 times faster than OCDD. Furthermore, it was suggested by Crosby, et al[91] that the presence of the high level of PCP in petroleum distillate will absorb the UV light before it can degrade the OCDD since the absorbance of PCP is approximately three times greater than OCDD at all UV wavelengths. However, while this suggests a rate difference it doesn't preclude eventual breakdown of both.

TABLE 4.—Photolysis of a Thin Film of PCP-Hexadecane Solvent Floating on Water.

Irradiation (hours)	Recovered PCP %	Recovered Control %
0	100	100
0.5	80	
1.1	56	
2.0	33	
4.0	20	
7.0	10	100

Photo decomposition rates of (○) OCDD (2.2 mg/1.) and (●) TCDD (5mg/1.) in purified methanol under simulated sunlight (F40BL lamp). (Reference 92)

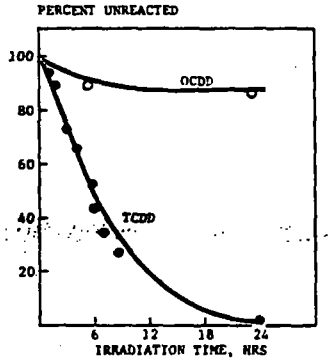


Figure 1.

Additional work on photolysis of PCP was done in the laboratory of Crosby and Wong.[93] Irradiation of a thin hexadecane film containing 400 ppm of PCP floating on the surface of deionized water in a closed flask resulted in very rapid loss of PCP (Table 4) but no detectable photodecomposition products. Most of the PCP was exposed in the floating film because only 3% of the PCP partitioned into the aqueous phase (pH 5.5) in three hours.

In deionized water, PCP (100 ppm) (I) was dechlorinated by light to 2,3,4,6- and 2,3,5,6-tetrachlorophenols, and chlorines were replaced by hydroxyl groups to give 3,4,5,6-tetrachlorocatechol, (II) tetrachlororesorcinol, and 2,3,5,6-tetrachlorohydroquinone. (III) When irradiated further, these broke down to dichlorocyclopentanediones (IV) and later to 2,3-dichloromaleic acid. (V)